



MARKED UP CLAIMS

41. A method of [treating] restoring NFκB activity in a mammal afflicted with an autoimmune disease resulting from a reduction in NFκB activity [in a mammal], comprising administering to a mammal suspected of suffering from said autoimmune disease a[n] therapeutically effective amount of agent which restores NFκB activity [in an amount and for a time sufficient to result in normal NFκB activity] 50 hours to treat said disease in said mammal.
42. The method according to claim 41, wherein said agent is [selected from the group that consists of] a protein [and a nucleic acid that encodes said protein].
43. The method according to claim 42, wherein said protein is selected from the group consisting of [that includes a mutant- or wild-type] NFκB, NFκB p50, NFκB p52, [a competitor of IκB that does not bind NFκB p50 or] NFκB p65, [a mutant- or wild-type NFκB p65, tumor necrosis factor-α, E-selectin, I-cam, and V-cam, interleukin-2, interleukin-6, granulocyte colony-stimulating factor, interferon-β, Lmp2, Lmp7, a ubiquitin-activating enzyme (E1), a ubiquitin-conjugating enzyme (E2), a ubiquitin-ligase (E3), a protein kinase, a proteasome subunit and an antibody directed against one of the 240 kD and 200 kD human erythrocyte proteasome inhibitors, CF-2] and IκB.
- [44. The method according to claim 41, wherein said agent is selected from the group that consists of a ribozyme, an antisense RNA molecule, a DNA molecule that encodes a said ribozyme, and a DNA molecule that encodes a said antisense RNA molecule.]
- [45. The method according to claim 44, wherein said ribozyme or said antisense RNA molecule is directed against one of the 240 kD and 200 kD human erythrocyte proteasome inhibitors, CF-2 and IκB.]